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1,1-Dichloroethane (CASRN 75-34-3)

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Reference Dose for Chronic Oral Exposure (RfD)

0409

1,1-Dichloroethane; CASRN 75-34-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 1,1-Dichloroethane

File First On-Line 10/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12/01/1996

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3

Not available at this time.

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I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

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183273

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3

Not available at this time.

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II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3
Last Revised -- 12/01/1996

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Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

[EPA Documentation, Review and, Contacts](#)

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II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification -- C; possible human carcinogen

Basis -- Based on no human data and limited evidence of carcinogenicity in two animal species (rats and mice) as shown by an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas in female rats and an increased incidence of hepatocellular carcinomas and benign uterine polyps in mice.

II.A.2. Human Carcinogenicity Data

None

II.A.3. Animal Carcinogenicity Data

Limited. An NCI bioassay (1978a) provides limited evidence of the carcinogenicity of 1,1-dichloroethane in Osborne-Mendel rats and B6C3F1 mice. This is based on significant dose-related increases in the incidence of hemangiosarcomas at various sites and mammary carcinomas in female rats and statistically significant increases in the incidence of liver carcinoma in male mice and benign uterine polyps in female mice. The study is limited by high mortality in many groups; the low survival rates precluded the appearance of possible late-developing tumors and decreased the statistical power of this bioassay.

Technical grade 1,1-dichloroethane in corn oil was administered by gavage 5 days/week for 78 weeks to groups of 50 Osborne-Mendel rats/sex/dose. All surviving animals were necropsied following a 33-week observation period. Due to toxicity, dosing was not continuous (3 weeks on then 1 week off), making the TWAs for 5 days/week 382 and 764 mg/kg/day for low- and high-dose males and 475 and 950 mg/kg/day for low- and high-dose females, respectively. Both a vehicle and an untreated (not intubated) control group (20 rats/sex/group) were included in the study. A high incidence of pneumonia (approximately 80%) in all 4 groups of each sex was considered to be the cause for the low survival at termination of the study. Survival at 111 weeks was 30, 5, 4, and 8% in the untreated control, the vehicle control, the low-dose, and the high-dose male rat groups, respectively. Survival at termination for the female rat groups was 40, 20, 16, and 18% for the untreated control, vehicle control, low- and high-dose groups, respectively. In female rats there was a statistically significant positive dose-related trend in incidence of hemangiosarcomas (0/19 for matched vehicle controls, 0/50 for the low-dose group, and 4/50 for the high-dose group). The incidence of mammary gland adenocarcinomas (1/20 for the untreated group, 0/19 for the vehicle control group, 1/50 for low-dose, and 5/50 for high-dose groups) showed a statistically significant dose-related positive trend in those female rats surviving at least 52 weeks; tumor incidence was 0/16, 1/28 and 5/31 for vehicle control, low- and high-dose groups, respectively. (Tumor incidence at termination for the untreated control females surviving at least 52 weeks was not reported.) This bioassay was conducted before the life table tests were implemented, so results adjusted for mortality are not available. No mammary gland adenomas or hemangiosarcomas were observed in the dosed-male rats.

In the same NCI (1978a) study, groups of 50 B6C3F1 mice/sex/group were administered technical grade 1,1-dichloroethane in corn oil by gavage 5 days/week for 70 weeks. As in the rat study, the dosage pattern was 3 weeks on and 1 week off; the surviving animals were necropsied 13 weeks after the termination of dosing. The TWAs for 5 days/week for the low- and high-dose groups were 1442 and 2885 mg/kg/day for male and 1665 and 3331 mg/kg/day for female mice. Control groups, identical to those in the rat study and consisting of 20 mice/sex/group were also used. Survival at termination was 80, 80, 80, and 50% for the untreated control group, the vehicle control group, the low-, and high-dose females, respectively. In male mice survival was 35, 55, 62, and 32% in the untreated control group, the vehicle control group, the low-, and high-dose groups, respectively. An increased incidence of hepatocellular carcinoma in male mice was not statistically significant by either pair-wise or trend test (2/17 in the untreated control group, 1/19 in the vehicle control group, 8/49 in the low-dose and 8/47 in the high-dose groups). The incidence of hepatocellular carcinoma in male mice surviving at least 52 weeks was 1/19, 6/72, 8/48, and 8/32 in the matched vehicle control group, a pooled vehicle control group consisting of mice from this and identical controls from other concurrent experiments, and the low-, and high-dose groups, respectively; this positive trend was statistically significant. In female mice, liver carcinomas were reported in only the vehicle control (1/19) and the low-dose groups (1/47); no liver tumors were seen in the untreated controls or in the high-dose group. A statistically significant increase in benign uterine endometrial stromal polyps (4/46) was observed in high-dose females; these were not observed in any other group. A preliminary report of the NCI (1978a) study was published by Weisburger (1977).

II.A.4. Supporting Data for Carcinogenicity

To determine if 1,1-dichloroethane in drinking water could act as a tumor promoter or a complete carcinogen, Klaunig et al. (1986) exposed groups of 35 male B6C3F1 mice to 1,1-dichloroethane in drinking water at 0, 835, or 2500 mg/L for up to 52 weeks following a 4-week treatment with either drinking water containing 10 mg/L diethyl nitrosamine (DNA-initiated groups) or with deionized water (noninitiated groups). The investigators estimated that the approximate weekly dose of 1,1-dichloroethane was 3.8 mg/g/week (corresponding to 543 mg/kg/day) for the groups

exposed to 2500 mg/L. Upon sacrifice at the end of either 24 weeks (10 mice/group) or 52 weeks (25 mice/group) of promotion, all tissues were examined for gross pathologic lesions and histologic sections of the liver, kidneys and lungs were examined. Neither the initiated nor the noninitiated 1,1-dichloroethane-treated groups showed a significant increase in the incidence of liver or lung tumors compared with initiated or noninitiated controls, respectively. The authors concluded that 1,1-dichloroethane was not carcinogenic to mice and did not act as a tumor promotor following initiation with DENA. These conclusions may not be entirely justified, since the duration of the study may have been inadequate for the development of tumors in noninitiated 1,1-dichloroethane-treated animals. In addition, the incidence of liver tumors in DENA-initiated controls was 70% at 24 weeks and 100% at 52 weeks, and the number of tumors/mouse in DENA-initiated controls at these times was 3.00 and 29.30, respectively. Hence, an increase in tumors or decrease in latency in 1,1-dichloroethane-treated DENA-initiated animals would have to be marked in order to be detectable.

Milman et al. (1988) and Story et al. (1986) investigated the chlorinated ethanes and ethylenes to detect their potential tumor initiating or promoting effects in a liver foci assay in Osborne-Mendel rats. In this assay, 1,1-dichloroethane did not give positive results for initiation (with phenobarbital as promotor), or as a complete carcinogen when administered in the absence of initiation or promotion. Positive results were seen for promotion with DENA as initiator. The assumption that the liver foci seen in this type of assay are precancerous has not been validated.

When tested by plate incorporation in a desiccator (because of volatility) in the presence and absence of metabolic activation systems, 1,1-dichloroethane was reported to be mutagenic for *Salmonella typhimurium* TA1535, TA98, and TA100, but not to TA1537 (Ricchio et al., 1983; Mitoma et al., 1984). Negative results were reported for 1,1-dichloroethane in a cell transformation assay with BALB/c-3T3 cells, tested in the absence of an exogenous metabolic activation system in a sealed glass incubation chamber (Tu et al., 1985; Arthur D. Little, Inc., 1983). When tested in a similar manner in a DNA repair assay with hepatocyte primary cultures from rats or mice, 1,1-dichloroethane produced positive results (Williams, 1977). The results obtained in these three assays were also summarized in a joint publication (Milman et al., 1988).

Positive results were obtained in a viral transformation assay in which 1,1-dichloroethane was incubated with cultured Syrian Hamster embryo cells in a sealed glass chamber prior to addition of adenovirus SA7 (Hatch et al., 1983).

Lattanzi et al. (1988) determined that 1,1-dichloroethane, like 1,2-dichloroethane, binds covalently to DNA, forming DNA adducts. The Covalent Binding Index (CBI) of both 1,1-dichloroethane and 1,2-dichloroethane classifies them as weak initiators.

Chronic bioassays performed by NCI (1978b) on the isomer 1,2-dichloroethane resulted in many of the same tumor types as seen in the bioassays of 1,1-dichloroethane. Significant increases in the incidences of forestomach squamous cell carcinomas and hemangiosarcomas were observed in male rats and an increased incidence of mammary adenocarcinomas was observed in both female rats and mice. In addition, alveolar and bronchiolar adenomas were reported in male and female mice; endometrial stromal polyps and sarcomas in female mice; and hepatocellular carcinomas in male mice.

Based on these findings, as well as the appearance of lung papillomas in mice after topical treatment, 1,2-dichloroethane was classified as a group B2 chemical, a probable human carcinogen (U.S. EPA, 1990). Because of similarities in structure and target organs, the carcinogenic evidence for 1,2-dichloroethane is considered to be supportive of the classification of 1,1-dichloroethane in group C, a possible human carcinogen.

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__II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

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__II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None.

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__II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

__II.D.1. EPA Documentation

Source Document -- U.S. EPA, 1985, 1986, 1990

The 1984 Health Effects Assessment for 1,1-Dichloroethane has received Office of Health and Environmental Assessment review.

__II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review -- 12/07/1989

Verification Date -- 12/07/1989

Screening-Level Literature Review Findings -- A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the cancer assessment for 1,1-Dichloroethane conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

__II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

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__III. [reserved]

IV. [reserved]

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VI. **Bibliography**

Substance Name -- 1,1-Dichloroethane
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VI.A. **Oral RfD References**

None

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VI.B. **Inhalation RfD References**

None

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VI.C. **Carcinogenicity Assessment References**

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Tu, A.S., T.A. Murray, K.M. Hatch, A. Sivak and H.A. Milman. 1985. In vitro transformation of BALB/c-3T3 cells by chlorinated ethanes and ethylenes. Cancer Lett. 28(1): 85-92.

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_VII. Revision History

Substance Name -- 1,1-Dichloroethane
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Date	Section	Description
08/01/1989	I.A.	Oral RfD now under review
05/01/1990	I.B.	Inhalation RfC now under review
10/01/1990	II.	Carcinogen assessment on-line
10/01/1990	VI.	Bibliography on-line
11/01/1991	ALL	FILES AD
01/01/1992	IV.	Regulatory Action section on-line
08/01/1995	I.A., I.B.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the

		workgroup functions beginning in September, 1995.
12/01/1996	II.D.3.	Primary contact removed
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
12/03/2002	II.D.2.	Screening-Level Literature Review Findings message has been added.

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VIII. Synonyms

Substance Name -- 1,1-Dichloroethane
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Last Revised -- 10/01/1990

75-34-3
AETHYLIDENCHLORID [GERMAN]
CHLORINATED HYDROCHLORIC ETHER
CHLORURE D'ETHYLIDENE [FRENCH]
CLORURO DI ETILIDENE [ITALIAN]
1,1-DICHLOORETHAAN [DUTCH]
1,1-DICHLORAETHAN [GERMAN]
1,1-DICHLORETHANE
DICHLORO-1,1 ETHANE [FRENCH]
1,1-DICHLOROETHANE
1,1-DICLOROETANO [ITALIAN]
1,1-DICLOROETANO [SPANISH]
ETHANE, 1,1-DICHLORO-
ETHYLIDENE CHLORIDE
ETHYLIDENE DICHLORIDE
HSDB 64
NCI-C04535
RCRA WASTE NUMBER U076
UN 2362

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